American Journal of Hematology 59:161–167 (1998)

Follicular Dendritic Cell Sarcoma and Interdigitating Reticulum Cell Sarcoma: A Review

R. Fonseca,[1] M. Yamakawa,[2] S. Nakamura,[3] P. van Heerde,[4] M. Miettinen,[5] T.W.H. Shek,[6] O. Myhre Jensen,[7] M.C. Rousselet,[8] and A. Tefferi[1]*

[1]Division of Hematology and Internal Medicine, Mayo Clinic and Mayo Foundation, Rochester, Minnesota
[2]Second Department of Pathology, Tokyo Women's Medical College, Tokyo, Japan
[3]Department of Pathology and Clinical Labs, Aichi Cancer Center Hospital, Nagoya, Japan
[4]Department of Cytology and Pathology, Het Nederlands Kanker Instituut, Amsterdam, The Netherlands
[5]Department of Pathology and Cell Biology, Thomas Jefferson University, Philadelphia, Pennsylvania
[6]Department of Pathology, University of Hong Kong, Queen Mary Hospital, Hong Kong, China
[7]Patologisk Institut, Århus Universitetshospital, Århus C, Denmark
[8]Department of Anatomical Pathology, Centre Hospitalier Universitaire, Angers Cedex 01, France

Neoplasms of reticular dendritic origin are extremely rare and include the follicular dendritic cell sarcoma (FDCS) and the interdigitating reticulum (or dendritic) cell sarcoma (IDCS). In this article, we review the literature pertaining to the two diseases and describe clinical observations and salient pathologic features, including information provided by authors of FDCS and IDDCS reports. We performed a computerized database search for published articles regarding FDCS and IDDCS. The articles were evaluated critically by the authors. Simple descriptive statistics were used to analyze the data. There are 51 cases of FDCS and 21 cases of IDDCS that are well documented in the literature. The pathologic diagnosis of FDCS and IDDCS is often challenging and requires morphologic, immunophenotypic, cytochemical, and electron-microscopic analysis. Patients with FDCS usually present with cervical or axillary lymphadenopathy, but extranodal disease has been described. In at least some patients, preexisting Castleman's disease has been recognized. Resected localized disease may be prevented from recurrence by consolidative radiotherapy. Chemotherapy regimens have shown nondurable antitumor activity in FDCS. Patients with IDDCS usually present with lymphadenopathy. The clinical course of IDDCS has been variable, but it seems to be more aggressive than that of FDCS. Variable degrees of remission may be achieved with chemotherapy. FDCS and IDDCS are rare neoplasms that may pose difficulty in pathologic diagnosis. IDDCS seems to display a more aggressive behavior than FDCS. Patients with IDDCS and FDCS can eventually die of disease progression. The role of chemotherapy and radiotherapy is not clearly defined. Am. J. Hematol. 59:161–167, 1998. © 1998 Wiley-Liss, Inc.

Key words: antigen-presenting cells; dendritic cells; drug therapy; lymphoproliferative disorders; neoplasms; sarcoma

INTRODUCTION

Neoplasms of reticular dendritic origin are extremely rare and include the follicular dendritic cell sarcoma (FDCS) [1–14] and the interdigitating reticulum (or dendritic) cell sarcoma (IDDCS) [2,15–30]. In the literature, 51 cases of FDCS and 21 cases of IDDCS have been well documented. This article reviews the literature pertaining to these two diseases and describes clinical observations and salient pathologic features. This description is particularly relevant because these neoplasms may mimic other lymphoproliferative disorders in their clinical presentation and morphologic appearance.

The main purpose of this article is to focus on available evidence regarding clinical features and therapeutic interventions in patients with FDCS and IDDCS. For

*Correspondence to: Ayalew Tefferi, M.D., Division of Hematology and Internal Medicine, Mayo Clinic, 200 First Street SW, Rochester, MN 55905.

Received for publication 29 May 1998; Accepted 10 June 1998

further pathologic description, the reader is referred to the original articles [1–31]. We also combine the information provided by authors of FDCS and IDDCS reports to provide extended follow-up and additional clinical information regarding these diseases.

MATERIAL AND METHODS

We performed a computerized database search for published articles regarding FDCS and IDDCS. The references of those articles were reviewed for additional information. Most authors of the articles were contacted to obtain updated and expanded information regarding the salient clinical and pathologic features of the patients. Additional follow-up information was obtained from several of them (M.Y., S.N., P.V.H., M.M., T.W.H.S., O.M.J., M.C.R).

Simple descriptive statistics were used to describe the group of patients. Using the data provided by the investigators and data obtained through published reports, we constructed a survival curve that was analyzed according to the method of Kaplan and Meier.

RESULTS

Follicular Dendritic Cell Sarcoma

Follicular dendritic cells. Four different types of dendritic (reticulum) cells exist in lymph nodes: histiocytic, fibroblastic, interdigitating, and follicular. Follicular dendritic cells (FDCs) participate in the immune system by presenting and retaining antigens for B cells and stimulating B-cell proliferation and differentiation. In addition, FDCs are known to have complex interactions with T cells.

FDCs are localized in the germinal centers of lymphoid follicles, in close contact with follicular center cells to which they present immune complexes (antigen-antibody). Normal FDCs are large (70 to 100 μm), often contain two nuclei, possess long cytoplasmic processes, and form clusters with lymphocytes. FDCs interact among each other, giving the characteristic appearance of giant cells. They attach to each other via desmosomes, which are unique to FDCs and are not seen in interdigitating dendritic cells (IDDCs). On electron microscopy, the presence of desmosomes is characteristic of FDCs. The cell of origin of FDCs is controversial. The coexpression of CD45 (leukocyte common antigen) and CD14 (monocyte antigen) has suggested the possible hematopoietic origin of FDCs. However, electron microscopic studies in rabbits have shown progression from fibroblastic reticulum cells.

Accurate immunostaining of FDCs has been difficult because of the proximity of these cells with lymphocytes. Positive immunostaining with the monoclonal antibodies R4/23 (murine IgM κ monoclonal antibody), Ki-FDC1p, and Ki-M4 (immunoglobulin [Ig]G-specific anti-lysosomal monoclonal antibody) is relatively specific to FDCs and has been demonstrated in both normal and clonal FDCs. In addition, FDCs may or may not stain positive for HLA-DR, which also is expressed in activated T lymphocytes, B cells, and monocytes. Expression of S-100, usually thought to be absent in FDCs, has been positive when determined by immunoperoxidase staining with the avidin-biotin complex, a more sensitive method. Other nonspecific markers expressed in normal FDCs include surface complement receptors (including CD35 [CR1], CD21 [CR2], CD11b [CR3]), lymphoid markers (CD19, CD20, CD21, CD23, CD24), and myeloid markers (CD14). Positive staining for CD45 (leukocyte common antigen), presumably seen in cells of hematopoietic origin only, has been reported by some investigators and refuted by others. Obviously, staining for any other antigen acquired in the process of antigenic presentation can be seen.

Pathologic features. The pathologic diagnosis of FDCS often is challenging and requires morphologic, immunophenotypic, cytochemical, and, occasionally, electron microscopic analysis. The neoplastic cells of FDCS display the characteristic spindle shape and multinucleation of normal FDCs [2,6] (Fig. 1). On electron microscopy, the long cytoplasmic projections and desmosomal junctions are noted. FDCS cells demonstrate staining characteristics similar to those of normal FDCs with certain antibodies [2,7,12,13], including R4/23 (5/8 tested, 63%), Ki-M4 (16/17, 94%), Ki-FDC1p (7/8, 88%), and CD35 (39/44, 89%) (Table I). In addition, CD21, a B-cell antigen present in follicular center cells, has been reported in nearly all cases (41/44, 93%). Other described markers include HLA-DR (4/7, 57%), CD45 (6/28, 21%), S-100 (13/42, 31%), vimentin (14/23, 61%) [7], EMA (7/17, 41%), and Ki-67 (5% to up to 50%). The reader is referred to the original sources for a more detailed pathologic description. It should be stressed that even though occasional lesions positive for CD19 and CD20 have been described, the vast majority of them are not and tumors do not display molecular evidence of a lymphoid origin.

As has been reported previously by experienced pathologists, the morphologic characteristics are such that the diagnosis can be entertained on routinely stained section [12]. Immunophenotyping usually is confirmatory, although there is significant overlap with IDDCS. Markers that are used to distinguish the two entities include those present in FDCS cells only (R4/23, CD21, CD35, complement receptors, 5′-nucleotidase activity) and those unique to IDDCS cells (ATPase activity). However, both FDCS and IDDCS cells may express HLA-DR, vimentin, and Ki-67. Conventional S-100 staining is positive in IDDCS and negative in FDCS. Epitope retrieval might explain the reported differences in staining positivity for S-100 in selected cases of

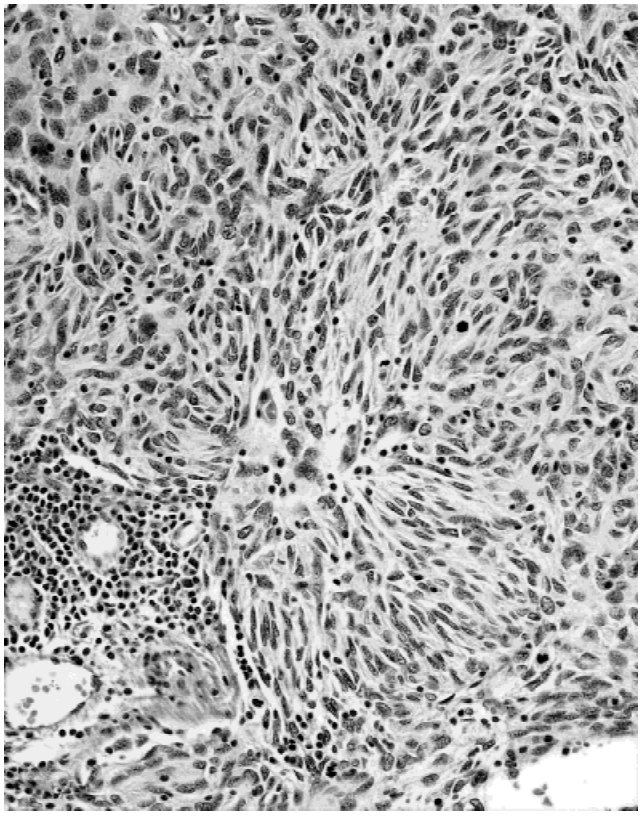

Fig. 1. Tissue section showing the typical pathologic features of a FDCS. The characteristic spindle cell morphology, along with a storiform, whorling pattern, is shown. (Hematoxylin-eosin.) (Courtesy of Professor Olaf Myhre Jensen.)

TABLE I. Results of Immunohistochemical Staining in Patients With FDCS and IDDCS*

	FDCS			IDDCS		
		Positive			Positive	
	Tested	No.	%	Tested	No.	%
CD1a	7	0	0			
CD11b				2	1	50
CD11c	3	0	0	5	5	100
CD14	4	3	75	7	4	57
CD19	3	2	67	5	1	20
CD20	25	1	4	10	1	10
CD21	44	41	93	7	0	0
CD22	5	1	20	3	0	0
CD30				4	2	50
CD35	44	39	89	7	0	0
CD45	28	6	21	15	10	67
CD45RO				5	3	60
R4/23	8	5	63	4	0	0
Ki-M4	17	16	94			
Ki-FDC1p	8	7	88			
ATPase				9	9	100
Vimentin	23	14	61	5	5	100
S-100	42	13	31	20	20	100
Cam 5.2	20	0	0			
AE1/AE3	7	0	0			
HLA-DR	7	4	57	12	11	92
EMA	17	7	41	3	0	0
Desmin				3	0	0
HMB 45	7	0	0			

*FDCS, follicular dendritic cell sarcoma; IDDCS, interdigitating dendritic cell sarcoma.

FDCS. When the avidin-biotin complex technique is used, S-100 staining has been reported to be positive in FDCS also [10,12,14]. When electron microscopy is available, the presence of desmosomal junctions differentiates these tumors from IDDCS.

Clinical course. Monda et al. [6] originally described FDCS in 1986. FDCS is a malignant neoplasm composed of FDCs [1–14]. The disease usually presents with cervical or axillary lymphadenopathy. However, extranodal disease has been described in at least 17 reported cases of FDCS. Some of the extranodal sites involved include the oral cavity, spleen, liver, small bowel, pancreas, and peritoneum. Constitutional symptoms are rare (5/51, 10%). Median age at presentation was 41 years (range, 17 to 76), and there was no sex predilection. Clinical course was variable; some patients enjoyed a prolonged and indolent clinical course, whereas others died as a consequence of disease progression.

Treatment. Reported treatments have been variable, and some of the treatment decisions were influenced by an initial misdiagnosis of FDCS as malignant fibrous histiocytoma or non-Hodgkin's lymphoma [2,7,8,14]. Information regarding the role of chemotherapy and radiation therapy is scarce. Because of the retrospective nature of the published reports, it is difficult to draw firm conclusions on which to base treatment recommendations. Thirty-one patients underwent surgical resection alone as primary treatment; 12 of these had relapse and required salvage treatment. Eight patients received radiation in combination with operation; six are alive and disease-free (median, 36 months; range, 7 to 66 months). These include a patient who received adjuvant radiation (5,400 cGy) to a completely resected involved field and remained alive and disease-free for 27 months [2] and another patient who received adjuvant radiation for incomplete resection and remained alive and disease-free for 12 months [12]. Two patients had disease recurrence after operation and radiation [14].

Twelve patients were treated with chemotherapy, and follow-up information is available for nine: four are alive and disease-free, three died of disease, and two are alive with disease. The reasons for administration of chemotherapy included bulky tumor [32], incomplete surgical resection [3,12], and an original diagnosis of non-Hodgkin's lymphoma [6,10]. The agents used consisted mostly of combination chemotherapy designed for the treatment of non-Hodgkin's lymphoma [14]. The patient we described previously [10] had a partial response to chemotherapy with CHOP (cyclophosphamide, hydroxydaunomycin [doxorubicin], vincristine [Oncovin], and

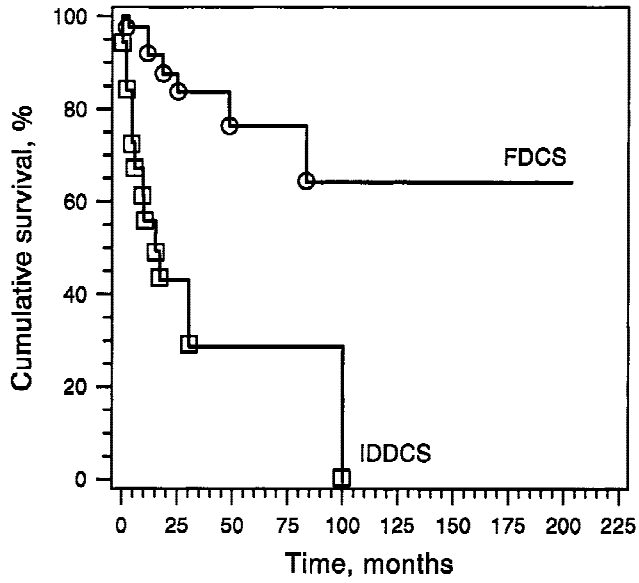

Fig. 2. Overall survival from time of original diagnosis (Kaplan-Meier) for patients with FDCS and IDDCS.

prednisone), had a further reduction in tumor bulk in response to chemotherapy with DHAP (dexamethasone, high-dose cytarabine [Ara-C] and cisplatin) but failed treatment with claridribine (2-CDA). A patient with recurrent disease after excision, reported by Monda et al. [6], received five courses of combination chemotherapy consisting of bleomycin, doxorubicin, cyclophosphamide, vincristine, and prednisone. The patient experienced a recurrence two years later. At the time of relapse, the patient was re-treated with an unspecified chemotherapy agent but failed to respond and died shortly thereafter. Another patient reported by the same authors received four cycles of chemotherapy consisting of doxorubicin, vincristine, and prednisone but was lost to follow-up.

In summary, localized disease either completely or incompletely resected can be prevented from recurrence by consolidative radiotherapy. More experience is needed to identify active chemotherapeutic agents for the treatment of disseminated FDCS. Chemotherapy regimens designed for the treatment of malignant lymphoma have shown nondurable antitumor activity in FDCS.

Survival. The duration of follow-up and other information was available for 42 patients. The median follow-up time was 22 months (range, 2 to 204 months). The median overall survival (Kaplan-Meier) from the time of diagnosis for the group has not been reached (Fig. 2). Of the 51 reported patients with FDCS (median follow-up, 22 months), 27 were alive and disease-free, 11 were alive with disease, 7 died of disease, and in six patients, follow-up information was not available.

Interdigitating Dendritic Cell Sarcoma

Interdigitating Dendritic Cells. Similar to FDCs, IDDCs also participate in the immune system as antigen-presenting cells, stimulating T lymphocytes. IDDCs normally are localized in the T-cell-rich areas of lymph nodes. IDDCs are believed to derive from hematopoietic precursors and belong to the mononuclear phagocytic system. Morphologically, IDDCs have a folded nucleus, in contrast to FDCs, which appear to have an elongated nucleus [19]. On electron microscopy, IDDCs have long and fine cytoplasmic extensions that intermingle with similar cells. In contrast to FDCs they lack desmosomes, and in contrast to Langerhans' cells they lack Birbeck granules.

IDDCs express CD45, HLA-DR, and S-100. In contrast to FDCs, they lack complement receptors and are negative for R4/23. In addition, IDDCs are strongly positive for ATPase [19], whereas FDCs are negative. FDCs are positive for 5-nucleotidase, whereas IDDCs are not. In contrast to Langerhans' cells, IDDCs do not express CD1.

Pathologic features. IDDCS grows in a whorl pattern of spindle-shaped cells [16] (Fig. 3). On electron microscopy, the cells show long cytoplasmic finger-like projections and bizarre nuclear shape [29]. Although desmosomes have been reported previously, they are characteristically absent [18]. No Birbeck granules should be observed because they are exclusive of Langerhans' cells. Malignant fibrous histiocytoma has been confused with IDDCS because of its spindle cell morphology. The reader is referred to the original sources for a more detailed pathologic description.

IDDCS has immunophenotypic characteristics similar to normal IDDCs, including strong ATPase activity and uniform positive staining for CD11c, vimentin, and S-100 (Table I). HLA-DR has usually been positive (11/12 tested, 92%), and reactivity against CD45 has been reported in 10 of 15 cases tested. IDDCS is usually negative for CD35, CD21, and CD20. Reactivity against K1-67 has been reported in only two cases (20% and 40%). No T-cell gene rearrangements have been found [2].

Clinical course. There are 21 well-documented cases of IDDCS reported in the literature. The patients usually presented with lymphadenopathy [2,15–30], and a few had constitutional symptoms such as fever (4/21, 19%). The median age at presentation was 52 years (range, eight to 74 years), and there was a slight male predilection (15/21). The clinical course of the disease was variable, but it seemed to be more aggressive than that of FDCS. Bone marrow involvement was reported in three of 15 cases. At least five patients have had evidence of extranodal involvement at the time of presentation. Interestingly, two patients had a clinical presentation consistent with superior vena cava syndrome [17,24].

Treatment. Treatment has varied according to the clinical context of the affected patients. Three patients underwent surgical resection alone. Follow-up data are available for two of them, and both are alive and disease-

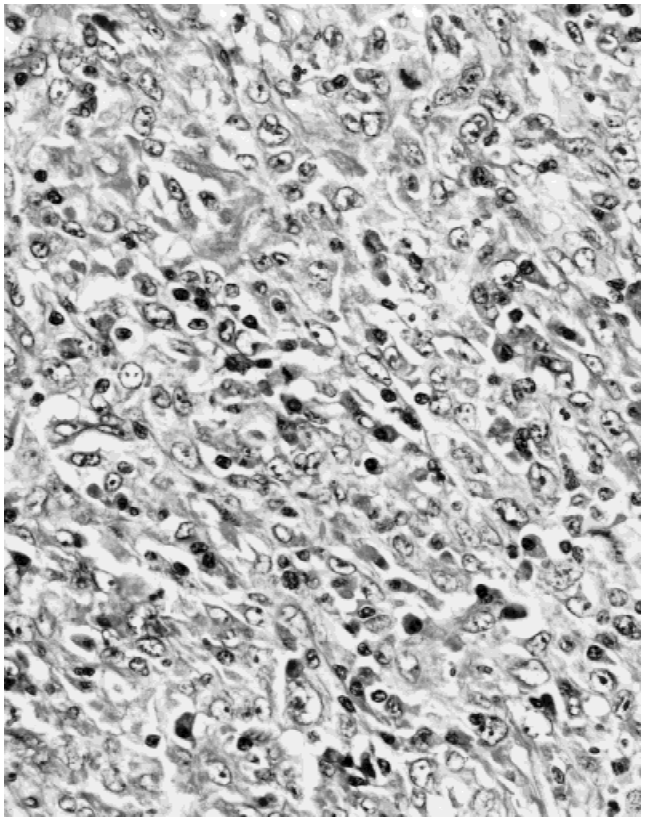

Fig. 3. Tissue section showing the typical pathologic features of an IDDCS. (Hematoxylin-eosin.) (Courtesy of Dr. Shigeo Nakamura.)

TABLE II. Chemotherapeutic Agents Used in the Treatment of Interdigitating Reticulum Cell Sarcoma and Response*

Reference	Chemotherapy regimen	Response
Nakamura et al. [18]	CHOP + radiation	CR
	CHOP?	Progression
Yamakawa et al. [27]	CHOP	PR
Miettinen et al. [25]	CHOP	CR
Rabkin et al. [23]	MACOP-B	PR
Salisbury et al. [22]	M-BACOD	Stable
Miettinen et al. [25]	M-BACOD	Progression
	Ara-C, P1, E	Progression
Turner et al. [19]	ABO	Progression
Chan and Zaatari [29]	BACO	Progression
Horschowski et al. [26]	P, Lasp, Vn, DN	Progression
Daum et al. [21]	POACEMcPr	PR
Rousselet et al. [24]	M-BACOD + radiation	PR
	MINE	PR
Feltkamp et al. [17]	CHOP + radiation	Progression
	CHOP	Progression
Hammar et al. [28]	COP + radiation	CR
Rabkin et al. [23]	COP + radiation	Progression

*A, Adriamycin (doxorubicin); Ara-C, cytarabine; B, bleomycin; C, cyclophosphamide; D, dexamethasone; DN, daunorubicin; E, etoposide; H, hydroxydaunomycin (doxorubicin); I, ifosfamide; Lasp, L-asparaginase; M, methotrexate; Mc, mechlorethamine; N, mitoxantrone; O, vincristine (Oncovin); P, prednisone; Pl, cisplatin; Pr, procarbazine; Vn, vindesine; CR, complete remission; PR, partial remission.

free [2,18]. One of these two patients received adjuvant radiation therapy after resection of a recurrence [2].

Three patients had radiation as their primary treatment, mostly for localized disease [16,18,19]. Two of them achieved a complete response and remain alive and disease-free [16,18]. One patient achieved a partial response and was alive with disease at last follow-up [19]. One patient had progressive disease and treatment had to be changed to salvage chemotherapy [16].

Ten patients received chemotherapy as primary treatment for their disease, mostly for advanced disease and systemic symptoms. All were evaluable for response. Four had progressive disease, two had stable disease, two achieved a partial response, and two achieved a complete response. Only one patient, in this last group, remained disease-free after 12 months of follow-up [25]. The chemotherapy regimens used were also of the type designed for the treatment of non-Hodgkin's lymphoma (Table II). Five patients underwent combined-modality treatment with chemotherapy and radiation; only one of them is alive and disease-free after 24 months.

Three patients underwent salvage high-dose chemotherapy with autologous bone marrow transplantation without much success; two patients were evaluable and both had recurrent disease [2,22,24].

In summary, the best management of localized disease appears to be similar to that of FDCS, which involves field irradiation. Variable degrees of remission may be achieved with chemotherapeutic agents used in malignant lymphoma. Remission durations are usually short, and the limited experience with bone marrow transplantation is not encouraging.

Survival. The duration of follow-up and other information was available for 19 patients. The median follow-up time was 12 months (range, one to 101 months). The median overall survival (Kaplan-Meier) from the time of diagnosis for the group was 15 months (Fig. 2). We could not estimate disease-free survival because information about time of relapse could not be obtained. Because of low numbers, no stratification was performed according to treatment administered. Of the 21 reported patients with IDDCS, 10 died of disease progression, two were alive with disease, six were alive and disease-free, one was lost to follow-up, and two died of unrelated causes (median follow-up, 12 months).

Differential Diagnosis of FDCS and IDDCS

The diagnosis of any one of these entities can rarely be entertained on clinical information alone. The presence of the disease may be suspected in patients with preexisting Castleman's disease or inflammatory pseudotumor, as has been previously reported. FDCS and IDDCS are rare and pose a diagnostic challenge to the surgical pathologist. It may be difficult, on morphologic review alone, to differentiate these diseases from each other and from other neoplastic processes, including Hodgkin's

disease, non-Hodgkin's lymphoma, soft tissue sarcomas, or nonneoplastic entities such as extranodal inflammatory pseudotumor. The use of immunohistochemical methods, electron microscopy, and molecular genetic studies usually helps to exclude Hodgkin's disease and non-Hodgkin's lymphoma.

Inflammatory pseudotumor, when present in extranodal locations, may be confused with FDCS. The morphology in inflammatory pseudotumor is characterized by a proliferation of spindle cells, along with the presence of inflammation and small vessel formation in the lymph node capsule, with extension along trabeculae of the node. The lack of morphologic atypia, lack of aggressive growth pattern, and the presence of a polymorphic cell population can distinguish it from FDCS. Unlike patients with FDCS, patients with inflammatory pseudotumor usually present with fever and other constitutional symptoms along with lymphadenopathy. There are reports of patients with inflammatory pseudotumor in whom FDCS developed later [9]; in one case, inflammatory pseudotumor was diagnosed during pregnancy and recurred six years later as FDCS. Similar to FDCS, inflammatory pseudotumor can present at extranodal locations, including lung, liver, and spleen. Most patients with intra-abdominal inflammatory pseudotumor present with pain, fever, and weight loss.

True histiocytic neoplasms are extremely rare, and many of those previously thought to be histiocytic tumors indeed represent malignant T-cell lymphoma, as determined by the finding of clonal rearrangements of the T-cell antigen receptors. These tumors can be differentiated from FDCS and IDDCS by their histiocyte-like appearance and by being negative for S-100. However, true histiocytic tumors should be included in the differential diagnosis with FDCS and IDDCS. Other entities that also need to be differentiated include histiocytosis X. Common disorders, which can be confused with either tumor, include sarcomas. Among these, special attention should be given in differentiating malignant fibrous histiocytoma, fibrosarcoma, and leiomyosarcoma, as well as spindle cell malignant melanoma. Anaplastic (Ki-1) CD30(+) non-Hodgkin's lymphoma has pleomorphic morphologic features that can make diagnosis difficult. It can present with a sarcomatous variant resembling other spindle cell neoplasms. Immunostaining for CD30, the associated t(2;5), and other morphologic features can distinguish this lymphoma from FDCS or IDDCS. FDCS or IDDCS initially diagnosed and treated as sarcomas or non-Hodgkin's lymphoma has been reported.

The intranodal myofibroblastoma, a rare neoplasm composed of spindled cells of stromal myoid origin, belongs in the differential diagnosis of FDCS or IDDCS. It almost always presents with lymphadenopathy of the groin, and the tumor has a distinct morphologic appearance with the presence of collagen deposition structures (the so-called amianthoid fibers).

CONCLUSION

FDCS and IDDCS are rare neoplasms that may pose difficulty in pathologic diagnosis. In several patients, a misdiagnosis of other neoplastic entities had been made before a final diagnosis of FDCS or IDDCS was made. Although patients can enjoy long-term survival after surgical resection, these tumors have the potential to recur. IDDCS seems to display a more aggressive behavior than FDCS. Patients with IDDCS and FDCS can eventually die as a result of disease progression. The role of chemotherapy and radiotherapy is not clearly defined. The use of radiation seems to prolong disease-free survival in patients undergoing resection. Although most patients respond to systemic chemotherapy designed for the treatment of malignant lymphoma, they all tend to have relapse. The ultimate treatment outcome seems to be more dependent on the stage at presentation and tumor biology.

REFERENCES

1. Hollowood K, Pease C, Mackay AM, Fletcher CD: Sarcomatoid tumours of lymph nodes showing follicular dendritic cell differentiation. J Pathol 163:205, 1991.
2. Weiss LM, Berry GJ, Dorfman RF, Banks P, Kaiserling E, Curtis J, Rosai J, Warnke RA: Spindle cell neoplasms of lymph nodes of probable reticulum cell lineage. True reticulum cell sarcoma? Am J Surg Pathol 14:405, 1990.
3. Chan JK, Tsang WY, Ng CS: Follicular dendritic cell tumor and vascular neoplasm complicating hyaline-vascular Castleman's disease. Am J Surg Pathol 18:517, 1994.
4. Shek TW, Ho FC, Ng IO, Chan AC, Ma L, Srivastava G: Follicular dendritic cell tumor of the liver. Evidence for an Epstein-Barr virus-related clonal proliferation of follicular dendritic cells. Am J Surg Pathol 20:313, 1996.
5. Chan JK, Tsang WY, Ng CS, Tang SK, Yu HC, Lee AW: Follicular dendritic cell tumors of the oral cavity. Am J Surg Pathol 18:148, 1994.
6. Monda L, Warnke R, Rosai J: A primary lymph node malignancy with features suggestive of dendritic reticulum cell differentiation. A report of 4 cases. Am J Pathol 122:562, 1986.
7. Pallesen G, Myhre-Jensen O: Immunophenotypic analysis of neoplastic cells in follicular dendritic cell sarcoma. Leukemia 1:549, 1987.
8. Hollowood K, Stamp G, Zouvani I, Fletcher CD: Extranodal follicular dendritic cell sarcoma of the gastrointestinal tract. Morphologic, immunohistochemical and ultrastructural analysis of two cases. Am J Clin Pathol 103:90, 1995.
9. Tanda F, Massarelli G, Cossu A, Bosincu L, Canu L: Follicular dendritic cell sarcoma following inflammatory pseudotumor in pregnancy (abstract). Int J Surg Pathol 2:S283, 1995.
10. Fonseca R, Tefferi A, Strickler JG: Follicular dendritic cell sarcoma mimicking diffuse large cell lymphoma: A case report. Am J Hematol 55:148, 1997.
11. Nayler SJ, Verhaart MJ, Cooper K: Follicular dendritic cell tumour of the tonsil. Histopathology 28:89, 1996.
12. Perez-Ordonez B, Erlandson RA, Rosai J: Follicular dendritic cell

tumor: Report of 13 additional cases of a distinctive entity. Am J Surg Pathol 20:944, 1996.
13. Nguyen DT, Diamond LW, Hansmann M-L, Hell K, Fischer R: Follicular dendritic cell sarcoma. Identification by monoclonal antibodies in paraffin sections. Appl Immunohistochem 2:60, 1994.
14. Chan JK, Fletcher CD, Nayler SJ, Cooper K: Follicular dendritic cell sarcoma. Clinicopathologic analysis of 17 cases suggesting a malignant potential higher than currently recognized. Cancer 79:294, 1997.
15. van Heerde P, Feltkamp CA, Feltkamp-Vroom TM, Koudstaal J: Sarcoma arising from interdigitating cells. Cytology and cytochemistry. Acta Cytol 27:306, 1983.
16. van den Oord JJ, de Wolf-Peeters C, de Vos R, Thomas J, Desmet VJ: Sarcoma arising from interdigitating reticulum cells: Report of a case, studied with light and electron microscopy, and enzyme- and immunohistochemistry. Histopathology 10:509, 1986.
17. Feltkamp CA, van Heerde P, Feltkamp-Vroom TM, Koudstaal J: A malignant tumor arising from interdigitating cells; light microscopical, ultrastructural, immuno- and enzyme-histochemical characteristics. Virchows Arch A Pathol Anat Histol 393:183, 1981.
18. Nakamura S, Koshikawa T, Kitoh K, Nakayama A, Yamakawa M, Imai Y, Ishii K, Fujita M, Suchi T: Interdigitating cell sarcoma: A morphologic and immunologic study of lymph node lesions in four cases. Pathol Int 44:374, 1994.
19. Turner RR, Wood GS, Beckstead JH, Colby TV, Horning SJ, Warnke RA: Histiocytic malignancies. Morphologic, immunologic, and enzymatic heterogeneity. Am J Surg Pathol 8:485, 1984.
20. Lennert K, Mohri N: Histopathology and diagnosis of non-Hodgkin's lymphomas. In Vehlinger E, ed. Malignant Lymphomas Other Than Hodgkin's Disease. Berlin: Springer-Verlag, 1978, p 448.
21. Daum GS, Liepman M, Woda BA: Dendritic cell phenotype in localized malignant histiocytosis of the small intestine. Arch Pathol Lab Med 109:647, 1985.
22. Salisbury JR, Ramsay AD, Isaacson PG: Histiocytic lymphoma: A report of a case with an unusual phenotype. J Pathol 146:99, 1985.
23. Rabkin MS, Kjeldsberg CR, Hammond ME, Wittwer CT, Nathwani B: Clinical, ultrastructural immunohistochemical and DNA content analysis of lymphomas having features of interdigitating reticulum cells. Cancer 61:1594, 1988.
24. Rousselet MC, Francois S, Croue A, Maigre M, Saint-Andre JP, Ifrah N: A lymph node interdigitating reticulum cell sarcoma. Arch Pathol Lab Med 118:183, 1994.
25. Miettinen M, Fletcher CD, Lasota J: True histiocytic lymphoma of small intestine. Analysis of two S-100 protein-positive cases with features of interdigitating reticulum cell sarcoma. Am J Clin Pathol 100: 285, 1993.
26. Horschowski N, Guitard AM, Arnoux I, Michel G, Thuret I, George F, Perrimond H: Interdigitating cell sarcoma: Occurrence during incomplete remission of a lymphoblastic lymphoma. Pathol Biol (Paris) 41: 255, 1993.
27. Yamakawa M, Matsuda M, Imai Y, Arai S, Harada K, Sato T: Lymph node interdigitating cell sarcoma. A case report. Am J Clin Pathol 97:139, 1992.
28. Hammar SP, Rudolph RH, Bockus DE, Remington FL: Interdigitating reticulum cell sarcoma with unusual features. Ultrastruct Pathol 15: 631, 1991.
29. Chan WC, Zaatari G: Lymph node interdigitating reticulum cell sarcoma. Am J Clin Pathol 85:739, 1986.
30. Vasef MA, Zaatari GS, Chan WC, Sun NC, Weiss LM, Brynes RK: Dendritic cell tumors associated with low-grade B-cell malignancies. Report of three cases. Am J Clin Pathol 104:696, 1995.
31. Chan JKC: Proliferative lesions of follicular dendritic cells: An overview, including a detailed account of follicular dendritic cell sarcoma, a neoplasm with many faces and uncommon etiologic associations. Adv Anat Pathol 4:387, 1997.
32. Selves J, Meggetto F, Brousset P, Voigt JJ, Pradere B, Grasset D, Icart J, Mariame B, Knecht H, Delsol G: Inflammatory pseudotumor of the liver. Evidence for follicular dendritic reticulum cell proliferation associated with clonal Epstein-Barr virus. Am J Surg Pathol 20:747, 1996.